

Risk and Resilience Factors for Posttraumatic Stress Symptomatology in Gulf War I Veterans

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What factors distinguish war-exposed veterans who experience posttraumatic stress symptomatology (PTSS) from those who do not? This study used structural equation modeling procedures to examine the complex interplay among predeployment, war-zone, and postdeployment factors as they relate to PTSS in a sample of Gulf War I veterans. A primary goal was to determine to what extent previously documented associations among Vietnam veterans would replicate in this more contemporary veteran cohort. Results supported a multivariate etiological perspective on PTSS, with war-zone factors accounting for the largest proportion of variance in PTSS. The majority of hypothesized associations held, suggesting that the mechanisms underlying PTSS may be similar across veteran cohorts.

Researchers have amassed evidence for a number of risk factors for posttraumatic stress disorder (PTSD) following trauma exposure (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003). However, most of the studies upon which this literature is based have focused on direct effects and used designs that do not address the likelihood that these factors may be related to PTSD through both direct and indirect pathways and via multiple causal chains (King, Vogt, & King, 2004; Vogt, King, & King, in press). This is unfortunate given that the use of single-

equation models and hierarchical, additive approaches may obscure important relationships among variables.

Although research findings point to the primacy of war-zone factors in accounting for PTSD among combat-exposed populations, a growing body of literature suggests the importance of extending investigations to examine predeployment and postdeployment factors. As researchers have noted, a stress reaction may be a consequence of a series of highly stressful life events that span the life course (King, King, Fairbank, Keane, & Adams,

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1998). Moreover, a number of factors that precede or follow stressful or traumatic events may increase or decrease risk for PTSD (Dohrenwend & Dohrenwend, 1981).

In the present study, we applied structural equation modeling (SEM) procedures to simultaneously examine relationships between predeployment, war-zone, and postdeployment risk and resilience factors and posttraumatic stress symptomatology (PTSS) in a cohort of U.S. veterans of the 1990–1991 conflict in the Persian Gulf region (GWI veterans). Although studies have documented associations between war-zone factors and PTSS among GWI veterans (e.g., Adler, Vaitkus, & Martin, 1996; Engel et al., 1993; Sutker et al., 1995; Wolfe, Proctor, Erickson, & Hu, 2002), to our knowledge, this is the first study to apply SEM to examine the complex network of explanatory factors for PTSS within this cohort.

The selection of risk and resilience factors was based largely on the prior work of King, King, and their colleagues (King et al., 1998; King, King, Foy, Keane, & Fairbank, 1999; King, King, Foy, & Gudanowski, 1996; King, King, Gudanowski, & Vreven, 1995) who applied SEM in a series of studies of risk and resilience factors for PTSD among Vietnam veterans. A primary goal was to determine the extent to which mechanisms identified in a more contemporary veteran cohort mirror those previously identified among Vietnam veterans. This study benefited from the use of a recently developed collection of deployment-related risk and resilience measures (Deployment Risk and Resilience Inventory [DRRI]; King, King, Vogt, Knight, & Samper, in press). The multifaceted nature and psychometric quality of these scales added a further dimension of depth to the current study. We also sought to expand our focus beyond statistical significance to a more in-depth consideration of the clinical significance of results, consistent with recent recommendations in the field (e.g., Harlow, Mulaik, & Steiger, 1997; Wilkinson & The APA Task Force on Statistical Inference, 1999). Although earlier work by King, King and their colleagues touched upon clinical significance, our intent was to provide an even more detailed examination of clinical significance as reflected in the strength of observed associations, both in

absolute terms and relative to one another (e.g., direct vs. indirect effects).

For the purpose of this study, we examined two predeployment, two war-zone, and two postdeployment factors. Hypotheses were drawn from King et al.'s (1999) findings and the supporting literature related to each category.

PREDEPLOYMENT FACTORS

Two predeployment factors were assessed: exposure to prior stressors and childhood family environment. Inclusion of the former stemmed from the recognition that exposure to prior stressors may influence reactions to subsequent stressors directly, in an additive manner, or indirectly, through the stressor's impact on other intervening factors. The direct explanation, which posits dual main effects of predeployment and war-zone factors and is based on Dohrenwend and Dohrenwend's (1981) additive burden model, has received empirical support in other military cohorts (Bremner, Southwick, Johnson, Yehuda, & Charney, 1993; Green, Grace, Lindy, Gleser, & Leonard, 1990; King et al., 1996; 1999). The indirect explanation posits that earlier stress experiences influence PTSD by increasing the likelihood of later stress exposure (Helzer, Robins, & McEvoy, 1987). This perspective is consistent with Hobfoll's loss spiral theory (Hobfoll, Dunahoo, & Monnier, 1995), suggesting that individuals who experience stress and trauma may lose resources that protect them from additional exposure.

The inclusion of childhood family environment was based on results indicating that individuals who experience positive childhood family environments encounter fewer later life stressors and have greater access to postdeployment social support (King et al., 1999). In turn, these factors may protect them against post-war symptomatology (Benotsch et al., 2000; King et al., 1998).

WAR-ZONE FACTORS

The GWI veterans were exposed to a number of war-zone circumstances that have been linked to PTSD (e.g., Adler, Vaitkus, & Martin, 1996; Engel et al., 1993; Sutker

et al., 1995; Wolfe et al., 2002), including traditional combat and the threat of immediate loss of life and long-term health consequences of potential environmental exposures (Norwood & Ursano, 1996). For the purpose of this study, two different conceptualizations of war-zone exposure were used: objective warfare exposure and subjective perception of threat (King et al., 1995). Previous findings indicate that the impact of warfare exposure on PTSD may be mediated through perceived threat (King et al., 1995), and this focus is consistent with the recent reformulation of PTSD (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition [DSM-IV]*; American Psychiatric Association, 1994) to require both objective and subjective aspects of trauma exposure.

POSTDEPLOYMENT FACTORS

Given ample evidence that individuals who experience additional postdeployment life stressors are vulnerable to PTSD (King et al., 1998) and that individuals who report higher postdeployment social support are less likely to develop PTSD (Benotsch et al., 2000; King et al., 1998; Sutker et al., 1995), these factors were also included. Prior research suggests various avenues through which these factors may mediate the impact of war-zone experiences on PTSD. For example, individuals who experience higher war-zone exposure may be exposed to additional stressors in the postdeployment period that put them at increased risk for PTSD (King et al., 1998). On the other hand, excessive support demands on the part of war-exposed veterans may induce the withdrawal of social support, leaving these individuals more vulnerable to PTSD (King et al., 1999).

HYPOTHESES

Drawing from the King et al. (1999) findings with Vietnam veterans and a review of the broader literature, we proposed several hypotheses. Overarching Hypothesis 1 posited that the effects of predeployment factors on posttraumatic stress symptomatology (PTSS) would be primarily mediated via later factors. Specifically, Hypothesis 1a proposed that both

predeployment factors would demonstrate an indirect effect on PTSS through postdeployment stressors. Hypothesis 1b posited that the effect of a positive childhood family environment would be further mediated via an increase in postdeployment social support. Hypothesis 1c proposed that the effect of prior stressors would be further mediated via an increase in the severity of warfare exposure. Hypothesis 1d proposed that prior stressors would demonstrate an additional direct effect on PTSS.

Hypothesis 2 posited that exposure to warfare would demonstrate an indirect effect on PTSS via perceived threat. Overarching Hypothesis 3 posited further mediation of deployment factors via postdeployment factors. Specifically, Hypothesis 3a proposed that exposure to warfare would demonstrate an additional indirect effect on PTSS via postdeployment stressors. Hypothesis 3b proposed that perceived threat would be related to PTSS through postdeployment social support. Hypothesis 4 suggested that deployment risk and resilience factors would be most strongly related to PTSS, followed by postdeployment, then by predeployment factors.

METHOD

Participants and Procedure

Our sampling pool consisted of 495 GWI veterans from across the country who were solicited for an earlier study but were not interviewed due to a higher than expected response rate. These veterans were originally identified through records held by the Defense Manpower Data Center and the VA Gulf War Health Registry. Questionnaires were sent to potential participants via U.S. mail, and we employed Mangione's (1998) multistep method to optimize responses. Potential participants were first mailed a letter that explained the purpose of the study, assured confidentiality, emphasized the voluntary nature of participation, and otherwise conformed to standards for the protection of human subjects. This letter was followed by a survey package containing a collection of stressor and health outcome measures. Later, a reminder card was sent, followed by a remailing of the package to nonrespondents,

and then a final reminder card. Of 478 potential respondents, 17 could not be reached, 11 returned incomplete questionnaires, and 308 of the remaining veterans (64%) returned complete questionnaires.

Participants were 45 years old ($SD = 9.0$) on average, 74% were men, 35% identified themselves as ethnic minorities, and 74% were married. Participants were deployed from Active Duty (26%), Reserve (43%), and National Guard (31%) units, and represented all branches of the military. More than a third of the sample (40%) was still in the military.

Measures

Scales from the Deployment Risk and Resilience Inventory. The Deployment Risk and Resilience Inventory (DRRI) includes scales to assess 2 predeployment factors (prior stressors and childhood family environment), 10 features of the deployment (combat experiences, perceived threat, aftermath of battle, difficult living and working environment, sense of preparedness, nuclear, biological, and chemical exposures, concerns about life and family disruptions, deployment social support, sexual harassment, general harassment), and 2 postdeployment factors (postdeployment social support and postdeployment stressors). One or more of these scales can be used as stand-alone instruments or the full set of scales may be used in concert. Evidence is available for the internal consistency reliability, test-retest reliability, discriminant validity, discriminative validity, and criterion-related validity of DRRI scales (King et al., in press). Moreover, the application of a focus group methodology to inform both the conceptualization of risk and resilience factors and the generation of items is a real strength of the DRRI, contributing to the content validity of this suite of scales (Vogt, King, & King, 2004).

Given the focus of this study on how exposure to circumstances of warfare relates to posttraumatic stress symptomatology, we included two deployment scales: a sum of combat experiences and aftermath of battle to assess warfare exposure and perceived threat. Given our interest in factors that precede and follow warfare exposure, we also included the following scales: prior stressors, childhood family envi-

ronment, postdeployment stressors, and postdeployment social support. All scales contained 15 items except for the postdeployment stressors scale, which contained 17 items. Additional details regarding content domains within these constructs and estimates of internal consistency reliability are provided in Table 1.

The PTSD Checklist. The PTSD Checklist (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993) was used to assess posttraumatic stress symptomatology (PTSS). The 17 items are directly adapted from the *DSM-IV* (APA, 1994) to assess reexperiencing and intrusive thoughts/memories, avoidance and emotional numbing, and hyperarousal symptoms. Respondents were asked to think about the deployment event or events that were most disturbing and rate how much they were bothered by each symptom in the past 3 months. This well-regarded and widely used instrument for assessing posttraumatic stress symptomatology has demonstrated coefficient alphas greater than .95, is highly correlated with other measures of PTSD, including the well-accepted and widely used Clinician-Administered PTSD Scale ($r = .93$; Blake et al., 1990), and has demonstrated acceptable levels of discriminant validity relative to measures of other forms of psychopathology (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996; Forbes, Creamer, & Biddle, 2001; Ruggiero, Del Ben, Scotti, & Rabalais, 2003; Weathers et al., 1993). Coefficient alpha was .96 in the current sample.

Overview of Analyses

A measurement model composed of both causal and effect indicators of latent variables was first examined and tested for gender invariance. A series of structural models were then specified, and we examined several types of evidence to evaluate study hypotheses: (a) fit indices corresponding to the overall model, (b) path coefficients and corresponding critical ratios for direct effects, (c) Sobel's tests of mediation (Baron & Kenny, 1986; Sobel, 1982) for indirect effects, and (d) effect sizes corresponding to proposed direct and indirect effects.

Table 1. Variables in the Model

| Construct | Description of indicators | α |
|-------------------------------------|---|----------|
| Predeployment factors | | |
| Prior stressors | Single causal indicator—Number of stressful life events experienced prior to deployment | .74 |
| Childhood family environment | Cohesion—Extent to which family spends time together and engages in shared activities | .83 |
| | Accord—Extent to which there is harmony within the family; lack of fighting or arguing | .88 |
| | Closeness—Extent to which family members share an emotional bond | .81 |
| | Total scale | .92 |
| War-zone factors | | |
| Exposure to warfare | Single causal indicator—Sum of combat experiences and exposure to circumstances of aftermath of battle | .91 |
| Perceived threat | Immediate fear—Concerns about immediate dangers, fear of death, or being wounded | .87 |
| | Long-term fear—Concerns about longer-term health risks | .88 |
| | Total scale | .89 |
| Postdeployment factors | | |
| Postdeployment stressors | Single causal indicator—Number of stressful life events experienced after returning from deployment | .73 |
| Postdeployment social support | General reception—Reaction from the general public upon return | .94 |
| | Feeling understood—Perception that others empathize with, understand, and value the veteran's experiences | .67 |
| | Concrete support—Access to person or persons who will offer practical assistance or support when needed | .84 |
| | Total scale | .86 |
| Posttraumatic stress symptomatology | Single causal indicator—Severity of PTSD symptoms | .96 |

RESULTS

Measurement Model Testing

Prior to specifying a measurement model, items from the seven scales were rationally grouped to create multiple indicators of each latent variable. Creation of these parcels was based on an internal consistency approach for multifaceted constructs (Little, Cunningham, Shahar, & Widaman, 2002). These parcels and evidence for unidimensionality in the form of Cronbach's alpha estimates are provided in Table 1. Confirmatory factor analyses using the Satorra–Bentler correction for nonnormality (Chou, Bentler, & Satorra, 1991) provided support for the acceptability of these parcels with Root Mean Square Errors of Approximation (RMSEA; Steiger, 1990) approaching the .08 standard of good fit (Browne & Cudeck, 1993), Standardized Root Mean Square Residual (SRMR) values below the recommended maximum of .10 (Browne & Cudeck, 1993), Comparative Fit Index (CFI; Bentler, 1990) values above the recommended minimum of .90 (Byrne, 1994), and Steiger's corrected form (Steiger, 1990) of the goodness of fit indices (GFI; Jöreskog & Sörbom, 1993) approaching the .95 recommended minimum. Factor loadings for each proposed parcel also appeared satisfactory. All specified factor loadings were significant, with critical ratios well above 2.00. The 3-factor solution for childhood fam-

ily environment yielded factor loadings above .50 for all 15 items (mean of .71). The 2-factor solution for perceived threat yielded loading above .65 for 10 of the 15 items, with all items loading above .50 ($M = .69$). The 3-factor solution for postdeployment social support yielded factor loadings of .50 or higher for 13 of 15 items, with all items loading above .35 ($M = .68$).

We next examined a measurement model (Model 1) in which each manifest indicator was specified to load on its respective latent variable. Loadings and measurement errors for causal indicators were set at 1.0, with the exception of PTSS, for which values were fixed according to scale reliability and variance. An examination of skewness and kurtosis supported the normality of study variables (i.e., skewness < 2 and kurtosis < 7 ; West, Finch, & Curran, 1995). Therefore, maximum likelihood estimation was used. Although the χ^2 for the resulting model was significant ($p < .05$), this is common in larger samples (Browne & Cudeck, 1993), and indices based on the noncentral chi square (RMSEA and CFI) indicated good to very good fit of this measurement model. Results for this and subsequent models are presented in Table 2.

Given previous findings of variant models for male and female veterans (King et al., 1999), the model was next tested for gender invariance. Nested models constraining measurement parameters to be equivalent (i.e., factor loadings, errors associated with latent variables, and associations

Table 2. Model Testing Sequence and Goodness-of-Fit Indices

| Model | Model fit | | | | | | Comparison | | |
|---|-----------|-----------|-------|-----|------|------|------------|------------------------|---------------------------|
| | χ^2 | <i>df</i> | RMSEA | CFI | SRMR | AGFI | Models | χ^2_{diff} | <i>df</i> _{diff} |
| 1. Initial measurement model | 69.11* | 37 | .05 | .98 | .036 | .98 | | | |
| 2. Measurement model including gender as variable | 109.69* | 42 | .07 | .96 | .041 | .97 | | | |
| 3. Hypothesized structural model | 159.04* | 50 | .08 | .94 | .060 | .95 | 3vs.2 | 49.35* | 8 |
| 4. Model revision #1 (delete path from prior stressors to PTSS) | 161.45* | 51 | .08 | .94 | .059 | .95 | 4vs.3 | 2.41 | 1 |
| 5. Model revision #2 (add paths from perceived threat to postdeployment stressors and warfare exposure to PTSS) | 120.97* | 49 | .07 | .96 | .045 | .97 | 5vs.2 | 11.28 | 7 |

Note. PTSS = posttraumatic stress symptomatology.

* $p < .05$.

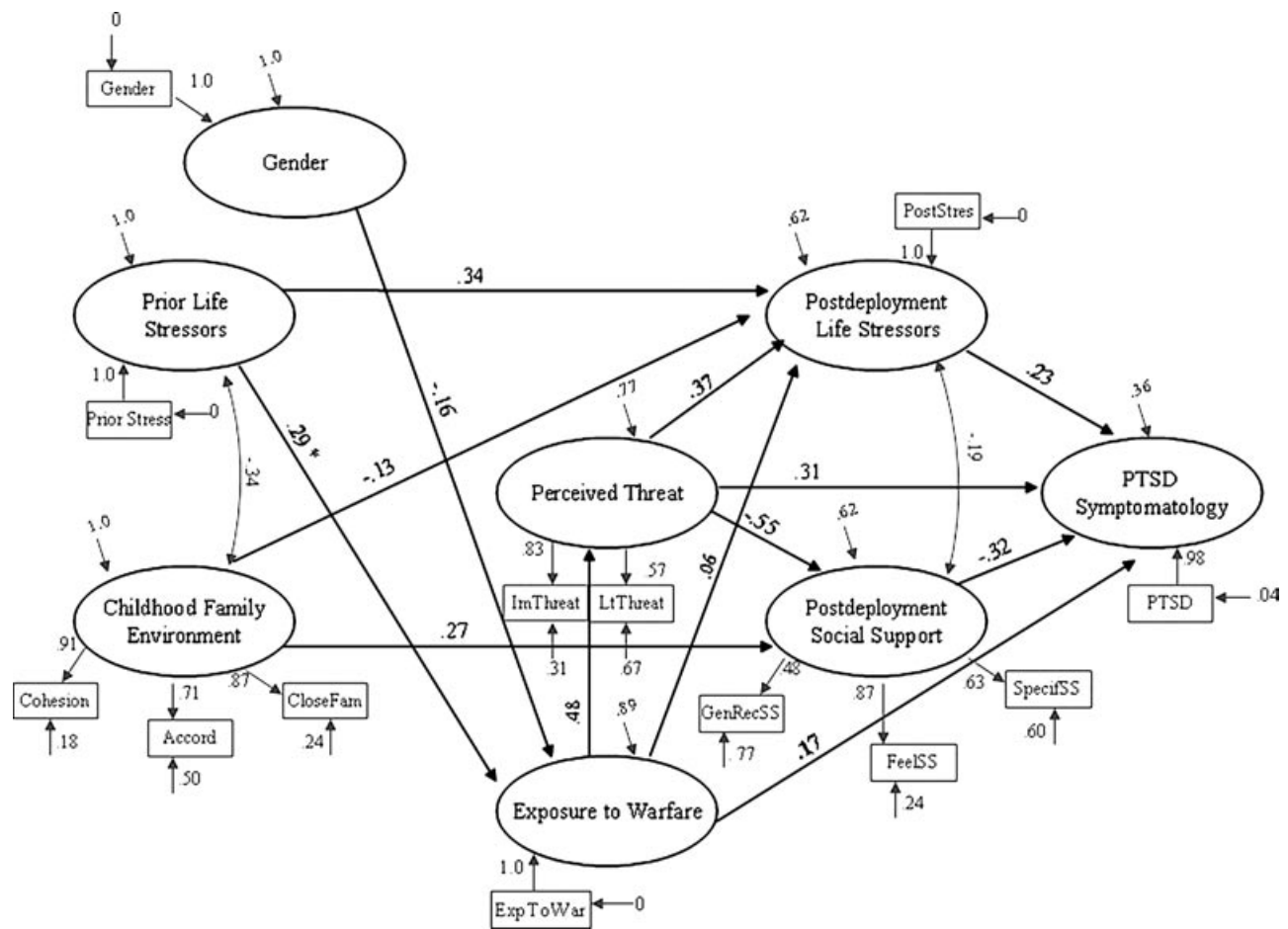


Figure 1. Final model incorporating both measurement and structural modeling results. Nonsignificant paths involving gender are omitted for ease of interpretation. Residuals associated with predeployment and postdeployment factors were allowed to freely covary within each category. All numbers represent standardized coefficients. Paths with bolded numbers were not initially predicted. All paths achieved statistical significance.

among latent variables) showed significantly worse fit than a model with freed parameters indicating that the factor structure of the variables was significantly different for men and women. However, the relatively small number of women in the current sample ($n = 81$) precluded testing separate structural models for the genders. Therefore, we utilized pooled within-gender factor means and covariances and integrated a dichotomous gender variable in further analyses to control for gender differences. As indicated in Table 2, the new measurement model incorporating this variable (Model 2) demonstrated acceptable fit. Parameter

estimates specific to the measurement model are included in Figure 1.

Structural Model Testing

We next examined a structural model that integrated all hypothesized paths, as well as paths from gender to each latent variable (Model 3). Error covariance was allowed within each set of predeployment and postdeployment variables. This model showed adequate fit to the data, but significantly damaged fit relative to a fully saturated structural

model (equivalent to the measurement model), suggesting only partial support for the full set of proposed hypotheses (See χ^2_{diff} , Table 2). We next examined coefficients and associated critical ratios for hypothesized paths. Findings provided support for all direct effects specified in Hypotheses 1 through 3, with the exception of the direct path from prior stressors to PTSS (Hypothesis 1d). Thus, we next estimated a second model in which this path was eliminated (Model 4) and evaluated the impact on model-to-data fit. As indicated in Table 2, deleting this path did not damage fit; therefore, we proceeded with a model in which this path was excluded.

Although fit indices for model 4 suggested adequate fit to the data, this model demonstrated significantly damaged fit relative to model 2, the fully saturated structural model, $\chi^2_{\text{diff}}(9) = 51.76$, $p < .05$. Thus, we reexamined the saturated model to identify additional, unhypothesized paths whose absence might be damaging fit. We identified two paths with corresponding t values larger than 3.0 and effect sizes suggesting clinically significant associations: paths from perceived threat to postdeployment stressors ($\beta = .37$) and from warfare exposure to PTSS ($\beta = .17$). The path from perceived threat to postdeployment stressors is consistent with Hobfoll's loss spiral theory (1995), suggesting that individuals exposed to stress and trauma may lose access to resources that protect them from subsequent stress exposure. The path from warfare exposure to PTSS is consistent with the idea that warfare exposure may exert an influence on PTSS that is either direct or

mediated through unstudied mechanisms. The addition of these paths resulted in a final model (Model 5) with a chi square statistic comparable to that of the fully saturated model (Model 2). This model, depicted in Figure 1, demonstrated good fit to the data. Both the RMSEA and SRMR were below minimum recommended values of .08 and .10, respectively, and the CFI and Steiger's corrected GFI were above recommended minimums of .90 and .95, respectively.

Evidence for hypothesized indirect effects was provided via the application of Sobel's tests of mediation (Baron & Kenny, 1986; Sobel, 1982). With two exceptions, results supported the statistical significance of all hypothesized indirect effects to PTSS: $z = 4.10$ for the indirect effect of prior stressors via postdeployment stressors; $z = -2.97$ for the indirect effect of childhood family environment via postdeployment social support; $z = 3.66$ for the indirect effect of warfare exposure via perceived threat; $z = 2.97$ for the indirect effect of perceived threat via postdeployment social support; $z = 3.29$ for the indirect effect of perceived threat via postdeployment stressors; $z = 2.83$ for the indirect effect of prior stressors via warfare exposure. Neither the indirect effect of childhood family environment via postdeployment stressors, $z = -0.14$, nor the indirect effect of warfare exposure via postdeployment stressors, $z = 1.43$, achieved statistical significance.

Then we next examined the strength of observed relationships to provide information that could be used to evaluate their clinical significance. Table 3 provides

Table 3. Parameter estimates for all effects on posttraumatic stress symptomatology (PTSS) from final model

| | Direct effects | | | Indirect effects | | | Total effects | | |
|-------------------------------|----------------|------------------------|------|------------------|------------------------|------|---------------|-------------------------|------|
| | Unstd. | Critical Ratio (z) | Std. | Unstd. | Critical Ratio (z) | Std. | Unstd. | Critical Ratios (z) | Std. |
| Prior Stressors | | | | 1.34 | 6.17 | .21 | 1.34 | 6.17 | .21 |
| Childhood family environment | | | | -0.55 | -3.86 | -.12 | -.55 | -3.86 | -.12 |
| Exposure to warfare | .43 | 3.45 | .17 | .74 | 6.05 | .29 | 1.17 | 9.08 | .46 |
| Perceived threat | .94 | 4.00 | .31 | .76 | 5.99 | .25 | 1.70 | 7.01 | .56 |
| Postdeployment stressors | 1.40 | 4.74 | .23 | | | | | 4.74 | .23 |
| Postdeployment social support | -11.29 | -4.43 | -.32 | | | | | -4.43 | -.32 |

Note. unstd. = unstandardized; std. = standardized; all z scores were significant at $p < .05$.

standardized estimates of all direct, indirect, and total effects of study variables on PTSS. Estimates of specific indirect paths are included below. Comparison of the influence of the two predeployment factors revealed that the effect of prior stressors on PTSS was nearly twice that of childhood family environment. Although prior stressors demonstrated indirect effects on PTSS via both war-zone and postdeployment factors, indirect effects mediated through warfare exposure ($\beta = .14$) were slightly larger than the indirect effect of prior stressors mediated through postdeployment stressors ($\beta = .08$). The effect of childhood family environment on PTSS was primarily mediated via postdeployment social support ($\beta = .09$) rather than postdeployment stressors ($\beta = .03, ns$), although this effect was still quite modest.

With respect to the influence of war-zone factors, perceived threat demonstrated a slightly stronger total effect than warfare exposure on PTSS. The direct effect of warfare exposure was modest, accounting for approximately one third of the total effect. The remaining two thirds of the effect was indirect, predominantly mediated via perceived threat ($\beta = .27$). Indirect effects of perceived threat via postdeployment social support ($\beta = .17$) were slightly stronger than via postdeployment stressors ($\beta = .09$), although both fell within the modest to moderate range. An analogous consideration of the relative contribution of effects via the two postdeployment factors revealed a slightly stronger total effect for postdeployment social support compared with postdeployment stressors.

Evidence pertaining to our final hypothesis was derived by summing the absolute values of each latent variable's standardized direct and indirect effects on PTSS across each category to quantify the influence of each set of factors. Consistent with Hypothesis 4, sums of the total effects for war-zone factors, postdeployment factors, and predeployment factors were 1.02, .55, and .33, respectively.

DISCUSSION

In the current study, we examined associations among predeployment, war-zone, and postdeployment risk and resilience factors and posttraumatic stress symptomatology

in GWI veterans. We benefited from the application of a sophisticated data analytic technique to examine complex pathways, as well as the availability of reliable and valid measures of key psychosocial risk and resilience factors. Furthermore, we attended to both the statistical and clinical significance of results. The latter was evaluated in terms of the strength of associations among variables (i.e., *r* values), although we duly note that there are occasions when small effects can be clinically significant and large effects relatively meaningless (Prentice & Miller, 1992).

Findings generally supported a multivariate etiological perspective. The set of risk and resilience factors accounted for 64% of the variance in PTSS. We found evidence for both direct and indirect effects, consistent with both Dohrenwend and Dohrenwend's (1981) additive burden model and Hobfoll's loss spiral theory (Hobfoll et al., 1995). Evidence for indirect effects via multiple pathways emphasizes the need for additional studies that acknowledge the complexity with which variables interrelate to predict posttrauma psychopathology (King et al., 2004; Vogt et al., in press). The relative contribution of risk and resilience categories was consistent with previous research with Vietnam veterans (Green et al., 1990; King et al., 1999; McTeague, McNally, & Litz, 2004), with war-zone factors accounting for the largest proportion of variance, followed by postdeployment, and then by predeployment factors.

The finding that all three sets of risk and resilience factors contributed meaningful variance to the prediction of PTSS highlights the importance of attending to events and circumstances that both precede and follow a focal trauma. In particular, the finding that both prior stressors and childhood family environment were related to PTSS, albeit through different mechanisms and to different degrees, suggests the importance of attending to these factors in future work. Similarly, the finding that warfare exposure demonstrated both direct and indirect effects via perceived threat supports the importance of attending to both objective and subjective aspects of trauma. Consistent with Vietnam veteran findings (King et al., 1996, 1999), the majority of the impact of warfare exposure was mediated via perceived threat. Results supported the relevance of factors

in the posttrauma environment, with a somewhat stronger role for social support than postdeployment stressors in mediating the effects of earlier factors on PTSS.

One of the major goals of hypothesis testing was to determine whether previously demonstrated associations for Vietnam veterans generalize to a more contemporary veteran cohort. Of 11 predicted paths, 10 were supported, and two additional unpredicted paths emerged. Thus, the majority of previously documented associations held, suggesting that the mechanisms underlying PTSS may be quite similar across these veteran cohorts. Although our final model supported many of our initial expectations based on prior work (King et al., 1996, 1999), there were notable exceptions. The finding that perceived threat was associated with exposure to additional stressors among GWI veterans but not Vietnam veterans is interesting. Although a number of other differences between this study and previous studies of Vietnam veterans (e.g., different measures, different lengths of time between exposure and assessment) preclude definitive conclusions, this finding may suggest a more prominent role for subjective aspects of warfare in predicting postdeployment circumstances for GWI veterans compared to Vietnam veterans. This finding is consistent with Hobfoll's conservation of resources theory (Hobfoll et al., 1995), indicating that individuals who experienced the highest levels of threat in the war-zone might be more vulnerable to subsequent stress exposures.

The finding that warfare exposure demonstrated a direct effect on PTSS implies the possibility of an additional unstudied mechanism through which objective aspects of warfare may have influenced PTSS in this cohort. Of course, support for both added paths should be considered preliminary given that they were included primarily on empirical grounds. As such, these findings may capitalize on chance (MacCallum, 1986), and replication is needed. Finally, in contrast with null results for Vietnam veterans (King et al., 1999), we hypothesized and found a relationship between prior stress exposure and warfare exposure. Given the use of the draft to enlist military personnel for the Vietnam War, prior experiences may have been less relevant in determining war-zone experiences for Vietnam veterans than for more recent cohorts.

A major limitation of the current study is the use of a cross-sectional design and dependence on retrospective self-reports. An excellent next step would be to combine the strength of SEM with a longitudinal design that allows for greater certainty regarding the directionality of relationships among key variables. In the present study, we cannot rule out a number of alternative explanations regarding the directionality of relationships. For instance, current symptoms might influence reporting of earlier life events. Although recent findings suggest this influence may not be as great as initially thought (King et al., 2000), this does not preclude the need for additional studies based on longitudinal designs.

Reliance on self-reports may also be problematic for reasons of method covariance (i.e., effects may be overestimated) and response bias (e.g., malingering). The use of other sources of data and other measures is recommended in future studies. Also useful would be studies that can examine separate models for men and women and explore potential interactions between risk and resilience factors. Finally, it should be noted that the current sample was assessed approximately 10 years after deployment. Given research findings indicating that risk factors for initial versus chronic PTSD may differ (e.g., Koenen, Stellman, Stellman, & Sommer, 2003; Schnurr, Lunney, & Sengupta, 2004), this study might therefore be best characterized as an investigation of risk and resilience factors for chronic rather than initial symptomatology.

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